UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF WASHINGTON
AT SPOKANE

NEIL HENRICKSEN and MAURITA HENRICKSEN, Plaintiffs, v. CONOCOPHILLLIPS COMPANY, Defendant.	NO. CV-07-224-JLQ MEMORANDUM IN SUPPORT OF PLAINTIFFS' CONSOLIDATED RESPONSE TO DEFENDANT'S VARIOUS MOTIONS TO STRIKE PLAINTIFFS' EXPERT WITNESSES AND MOTIONS FOR SUMMARY JUDGMENT

TO THE HONORABLE COURT:

Come now, Plaintiffs, Neil and Maurita Henricksen, and file this Memorandum in Support of Plaintiff's Consolidated Response to Defendant's Various Motions to Strike Plaintiffs' Expert Witnesses and Motions for Summary Judgment. To avoid unnecessarily burdening the

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Court with repetitive argument and authority, Plaintiffs submit this brief in response to the following motions:

1. Motion to Exclude Causation Opinions Based Upon Unreliable Epidemiological Evidence;

2. Motion to Exclude Plaintiffs' Expert Frank Gardner;

3. Motion in Limine to Limit the Testimony of Plaintiffs' Treating Physicians;

4. Motion to Exclude Plaintiffs' Expert Marco Kaltofen;

5. Motion to Exclude Plaintiffs' Expert William Sawyer

Also, because the same issues raised by these motions to exclude expert testimony are the bases for ConocoPhillips's Motion for Summary Judgment on General Causation and Motion for Summary Judgment on Specific Causation, this response will address ConocoPhillips' motions for summary judgment as well.

In support of this consolidated response, Plaintiffs would respectfully show the Court as follows.

I. Introduction

One of the few matters in science that has reached the level of virtual certainty is that exposure to benzene causes acute myelogenous

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leukemia ("AML"). There is no rational dispute of this point in science or in this case. As early as 1977, the Occupational Safety and Health Administration found that the scientific evidence "conclusively establish[ed] that benzene causes leukemia." BEN 112 at 170377.[1] ConocoPhillips' own corporate representative acknowledges that benzene causes AML. *See* Deposition of Jennifer Galvin at 234.

It is equally beyond dispute that gasoline contains benzene, *see* Deposition of Jennifer Galvin at 163, 168, and that Neil Henricksen was exposed to benzene while loading and unloading gasoline for ConocoPhillips Corporation ("ConocoPhillips"). While ConocoPhillips has conveniently destroyed most records of the benzene content of its gasoline during the time Mr. Henricksen was exposed to its product, ConocoPhillips' own Material Safety Data Sheets ("MSDS") indicate that ConocoPhillips' gasoline contained as much as 4.9% pure benzene. *See, e.g.,* CONCO 5004 at 1. Consequently, there is no dispute that Mr. Henricksen was exposed to benzene vapors while working with ConocoPhillips' gasoline, and it should come as no surprise that

[1] See accompanying Declaration of Counsel for all exhibits referenced herein.

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Plaintiffs' expert witnesses have reached the rational conclusion that Mr. Henricksen's AML was caused by this exposure.

Nevertheless, ConocoPhillips filed a series of motions to strike the testimony of Plaintiffs' expert witnesses, arguing that insufficient evidence supports both the general and specific causation opinions of Plaintiffs' experts. ConocoPhillips' contentions are based primarily on the absurd suggestion that there is no scientifically reliable proof that *gasoline* causes AML. ConocoPhillips' argument is little more than a cheap parlor trick intended to divert the Court's attention from the true nature of this suit, which is that Mr. Henricksen was exposed to substantial amounts of *benzene* from ConocoPhillips' gasoline. That ConocoPhillips has resorted to misdirection is indicative of its awareness that it has no defense against the universally accepted fact that the benzene to which Mr. Henricksen was exposed causes the very disease he contracted.

II. Factual Background

Contrary to ConocoPhillips' repeated mischaracterizations, this is a benzene exposure case. From 1976 to 1983, Mr. Henricksen transported gasoline and diesel fuel exclusively from a ConocoPhillips

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terminal by tanker truck to fuels stations in the Spokane, Washington area. *See* Deposition of Neil Henricksen at 217-18. Mr. Henricksen re-loaded his tanker truck at the ConocoPhillips terminal between 2 and 5 times each day. *Id*. at 194. On average, Mr. Henricksen worked 5 or 6 days a week in 10 to 12 hour shifts and loaded his tanker truck at the ConocoPhillips facility 25 to 30 times per week. *Id*. Approximately half of the fuel loads from the ConocoPhillips terminal were diesel and half were gasoline. *Id*. at 242-43.

During the entire time Mr. Henricksen transported fuel from the ConocoPhillips terminal, Mr. Henricksen drove a top-loading truck that was filled through a hatch in the top of the tank. *Id*. at 178-80. Unlike other terminals where Mr. Henricksen had worked, the ConocoPhillips terminal did not have a vapor recovery system to prevent exposure to the gasoline fumes. *Id*. at 181. Additionally, the ConocoPhillips terminal was not an "open" terminal such that fumes would easily dissipate. *Id*. at 178-81. Instead, it was a "closed" terminal with a roof and partial walls on two of the four sides similar to the terminal depicted in Exhibit 5. *Id*. In addition, Mr. Henricksen regularly spilled ConocoPhillips' gasoline on his skin. *Id.* at 192-96.

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In August 2003, Neil Henricksn was diagnosed with acute myelogenous leukemia at the age of 61. There is no dispute in the scientific community or this case that exposure to benzene causes AML, and there is no dispute that the ConocoPhillips gasoline that Mr. Henricksen loaded and unloaded for 8 years contained benzene. Consequently, this case is one of the rare toxic tort cases in which there is no dispute that the plaintiff was exposed to a toxin and contracted a disease that the toxin is known to cause.

III. Standard of Review

A. The *Daubert* Standard.

Admissibility of scientific evidence is governed by Federal Rule of Evidence 702. Under the Supreme Court's interpretation of Rule 702 in *Daubert*, expert testimony is admissible if it is relevant and reliable. *Daubert v. Merrell Dow Pharms., Inc.,* 509 U.S. 579, 587-89, 113 S.Ct. 2786 (1993) ("*Daubert I* "). Expert's opinion is relevant if the knowledge underlying it has a "valid ... connection to the pertinent inquiry." *Kumho Tire Co. v. Carmichael,* 526 U.S. 137, 149, 119 S. Ct. 1167 (1999) (quoting *Daubert I,* 509 U.S. 579, 592, 113 S. Ct. 2786). Scientific evidence is reliable if it is grounded in the methods of science. *Id.* at

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595-96,113 S. Ct. 2786. It is the role of the district court to serve as a "gatekeeper," excluding bad science that lacks sufficient indicia of reliability to be admissible. *Daubert v. Merrell Dow Pharms., Inc.,* 43 F.3d 1311, 1316 (9th Cir. 1995) ("*Daubert II* ").

Here, the only objection raised by ConocoPhillips is that the proffered testimony of Plaintiffs' experts is unreliable. In determining whether an expert's opinion is reliable, the focus is on the expert's principles and methodology, not the conclusions. *Id.* The Supreme Court provided four non-exclusive factors to consider as part of the reliability analysis:

1. whether the scientific theory or technique can be (and has been) tested;
2. whether the theory or technique has been subjected to peer review and publication;
3. whether a particular technique has a known potential rate of error; and
4. whether the theory or technique is generally accepted in the relevant scientific community.

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Daubert I, 509 U.S. at 593-94, 113 S. Ct. 2786. Because there are innumerable types of experts and expertise, these factors "may or may not be pertinent in assessing reliability, depending on the nature of the issue, the expert's particular expertise and the subject of his testimony." *Kumho Tire Co.,* 526 U.S. at 150, 119 S. Ct. 1167.

B. *Daubert* and the Right to a Jury Trial.

In performing a *Daubert* analysis, the Court must remain mindful that "*Daubert* makes the district court a gatekeeper, not a fact finder." *U.S. v. Sandoval-Mendoza*, 472 F.3d 645, 654 (9th Cir. 2006). When credible, qualified experts disagree, the parties are entitled to have jury determine whether the plaintiff has met its burden of proof. U.S. CONST. amend. VII; *Sandoval-Mendoza*, 472 F. 3d at 653. The role of the Court is to determine whether the proffered expert testimony is reliable "without interfering with the jury's role as trier of fact . . . ." *See* Stephen Breyer, *Introduction* to REFERENCE MANUAL ON SCIENTIFIC EVIDENCE at 4 (2nd ed. 2000).

Consequently, the gatekeeping function of the Court is not to determine which expert witness is correct. "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden

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of proof" remain the "appropriate means of attacking shaky but admissible evidence." *Daubert I*, 509 U.S. at 596, 113 S. Ct. 2786. As the Ninth Circuit has noted:

> A trial court's determination that the proffered testimony of one expert witness is reliable and helpful does not necessarily mean that *the contradictory testimony of another witness,* concerning the same subject matter but *using a different methodology,* is not also reliable and helpful.
>
> . . . .
>
> If two contradictory expert witnesses [can offer testimony that is reliable and helpful], both are admissible, and it is the function of the finder of fact, not the trial court, to determine which is the more trustworthy and credible.

Dorn v. Burlington Northern Santa Fe R.R., 397 F.3d 1183, 1195 (9th Cir. 2005) (quoting WEINSTEIN'S FEDERAL EVIDENCE § 702.05[3], at 702-80.12 to 702-80.13). Instead, "A court may admit somewhat questionable testimony if it falls within 'the range where experts might reasonably differ, and where the jury must decide among the conflicting views . . . .'" *S.M. v. K.M.*, 262 F.3d 914, 921 (9th Cir. 2001) (citing *Daubert,* 509 U.S. at 596, 113 S. Ct. 2786). In short, the Court "must respect the jury's constitutionally specified role" as the finder of facts. *See* Stephen Breyer, *Introduction* to REFERENCE MANUAL ON SCIENTIFIC EVIDENCE at 4 (2nd ed. 2000).

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IV. Plaintiffs' Experts are Highly Qualified Scientists

As an initial matter, it is worth noting that ConocoPhillips does not contend that Plaintiffs' experts lack the necessary qualifications to give the opinions offered in this case. This is most likely because the training and experience of these experts is unassailable and because ConocoPhillips cannot afford for the Court to question how such imminently qualified scientists could be so blatantly wrong in their scientific analysis that their opinions are not even admissible before a jury. Instead, as demonstrated below, *Daubert* and its progeny were never intended to preclude expert witness of this caliber from presenting to a jury the same opinions these experts would have in their scientific practices.

A. Peter Infante

Peter Infante is the managing member and President of Peter F. Infante Consulting, L.L.C., in Falls Church, Virginia. Dr. Infante received his Ph.D from the University of Michigan, Department of Epidemiology, in 1973. Soon after receiving his doctorate, Dr. Infante began his long and distinguished career as an Epidemiologic Consultant for the World Health Organization in Washington, D.C., and then continued his work

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as an epidemiologist in Ohio before becoming the Acting Chief of the Biometry Section of the National Institute for Occupational Safety and Health, Center for Disease Control, in Cincinnati, Ohio, where he focused on occupational epidemiological studies and investigations to determine associations between exposure to toxic substances and cancer, among other things.

In 1978, Dr. Infante moved to Washington, D.C., where he assumed the position of Director of the Office of Carcinogen Identification and Classification for the Department of Labor. From 1983 to 2002, he was the Director of the Office of Standards Review in the Occupational Safety and Health Administration. His was the primary agency responsible for reviewing existing OSHA standards and making recommendations for modifications based on risk assessments and epidemiologic, toxicologic, and industrial hygiene data. His office was also responsible for regulation of toxic substances in the workplace.

In 2002, Dr. Infante began his consulting firm where he consults in occupational and environmental health. Dr. Infante also serves as an Adjunct Professor of Environmental and Occupational Heath at The George Washington University.

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Dr. Infante has served in numerous advisory and consultant positions including serving as a member on the WHO Expert Committee on the Evaluation of the Carcinogenic Risk of Chemicals in Humans and the National Academy of Sciences Subcommittee to revise Emergency Exposure Guidance Levels for Benzene and Ethylene Oxide. Additionally, Dr. Infante has received several awards for his research, notably a special commendation from the National Institute for Occupational Safety and Health for research contributions toward understanding the toxicology of Benzene and Beryllium, and a Special Achievement Award from the U.S. Department of Labor.

Dr. Infante is a member of the American College of Epidemiology, American Conference of Governmental Industrial Hygienists, and American Public Heath Association. He is the author or co-author of numerous publications including Leukemia in Benzene Workers, Benzene and Leukemia, Benzene Toxicity: Studying a Subject to Death, and Health Effects of Gasoline Vapors: Benzene. Dr. Infante has spent most of his formidable career studying the health effects of Benzene exposure to humans and has become one of the nation's, if not the

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world's, leading authorities on the subject. *See* Curriculum Vitae of Dr. Peter Infante.

B. William Sawyer

Dr. William R. Sawyer is the Chief Toxicologist at Toxicology Consultants & Assessment Specialists, Inc., in Skaneateles, New York. Dr. Sawyer received his Ph.D. in toxicology from Indiana University School of Medicine in 1988. He began his distinguished career as the Chief Toxicologist of Onondaga County Department of Health in Syracuse, New York, where he was responsible for municipal and civil risk assessment, the evaluation of environmental exposures, and where he advised and communicated with the Office of the Environment/County executive and legislative subcommittees with respect to public health and environmental health issues. In 1993, Dr. Sawyer became the laboratory director of EXPRESSLAB, Inc. He also spent several years as the Associate Editor of *Practical Reviews in Forensic Medicine and Sciences*. In addition to acting as Chief Toxicologist for Toxicology Consultants & Assessment Specialists, Inc, Dr. Sawyer also currently serves as an assistant professor at SUNY Upstate Medical University and is a member of the Editorial Advisory Board for The Forensic

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Board for The Forensic Examiner.

Dr. Sawyer is board certified in forensic medicine, toxicology, and pharmacology. Dr. Sawyer has been a speaker at several seminars where he has given presentations on several topics regarding toxicology, including a presentation on "Evaluating Toxic Exposures after Daubert," "The Medical Aspects of Toxic Exposure Assessments," and "Fundamentals of Medical Toxicology." Dr. Sawyer has also authored or co-authored numerous publications, abstracts, treaties and editorial publications, including one entitled "A Fatal Case of Benzene Poisoning & Special Discussion by Drs. Sawyer & Rigle."

C. Marco Kaltofen

After obtaining his degree in civil engineering, Marco Kaltofen, P.E., began his career as a chemist for the New England Aquarium in Boston where he traced the environmental fate of petroleum drilling wastes and the fate of pollutants in the oceans. In 1984, Mr. Kaltofen became a project coordinator for Greenpeace International in London where he was responsible for environmental program research and field sampling. Four years later, Mr. Kaltofen founded the Citizens' Environmental Laboratory in Boston where he served as Laboratory

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Director, performing engineering and chemical quality evaluations of contaminated sites in addition to managing the nationally recognized nonprofit organization. Also in 1988 Mr. Kaltofen accepted the position of President of Boston Chemical Data Corporation where he provides technical support for environmentally-related organizations as well as performing environmental investigations onsite and via computerized chemical and engineering information systems. Mr. Kaltofen conducts extensive onsite investigations in the U.S. and internationally.

Mr. Kaltofen has realized many professional achievements throughout his accomplished career, including the review of industrial air monitoring data for total hydrocarbons and benzene, with calculation of human exposure to benzene as well as the design and completion of sampling efforts to complete indoor air quality studies for gasoline-related volatile organic compounds.

D. Frank Gardner

Frank Gardner received his M.D. from Northwestern University Medical School in Chicago, Illinois. In 1949, Dr. Gardner began his career as an Instructor in Medicine at Harvard Medical School and Associate in Medicine at Peter Bent Brigham Hospital, both in Boston.

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Two years later, he became an Associate Hematologist at Boston Lying-In Hospital and continued throughout the next several years to study, teach, and practice hematology as an Attending Hematologist, Consultant in Hematology and Professor of Medicine. In 1966, Dr. Gardner accepted the position of Director of the Hematology Research Laboratory at Presbyterian University of Pennsylvania Medical Center in Philadelphia, Pennsylvania. While there, he continued to serve as a Professor of Medicine and Consultant in Hematology. In 1969, Dr. Gardner became the Director of Medicine at Presbyterian until 1975 when he moved to Galveston, Texas to lead the Hematology-Oncology Division at The University of Texas Medical Branch ("UTMB") as its Director as well as a Professor of Medicine. In 1990, Dr. Gardner became a Clinical Professor of Medicine in the Division of Hematology-Oncology in Galveston where he continues to serve in this capacity. Dr. Gardener is board certified in Internal Medicine and licensed in several states.

Throughout his accomplished career, Dr. Gardner served as a board member or chairman of numerous committees and societies, including the American Society of Hematology, the National Cancer

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Institute, American National Red Cross, an the Biohazards Committee of UTMB. As an author or co-author, Dr. Gardner has been published in countless medical journals, his articles focusing primarily on diseases of the blood, diseases of the marrow, and the study and treatment of leukemia.

V. The Opinions of Plaintiffs' Experts Are Well Supported by Scientific Reasoning and Literature.

What is truly remarkable about this case is that there are so few disputed facts. Unlike the allegation in *Daubert* that Bendectin causes birth defects, there is absolutely no dispute that benzene causes AML. There is also no dispute that ConocoPhillips' gasoline contained benzene and that Mr. Henricksen was exposed to benzene from ConocoPhillips' gasoline. The only disputes that appear to exist are whether a plaintiff must prove that a particular source of benzene is capable of causing AML and whether the amount of benzene to which Mr. Henricksen was exposed was sufficient to cause his AML. As demonstrated below, neither dispute warrants the exclusion of testimony from Plaintiffs' experts.

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A. Proof of Causation in Toxic Tort Cases

Causation in a toxic tort case is generally discussed in terms of general and specific causation. *In Re Hanford Nuclear Reservation Lit.,* 292 F.3d 1124, 1133 (9th Cir. 2002). General causation refers to "whether the substance at issue had the capacity to cause the harm alleged." *Id.* Specific causation refers to whether a particular individual's injury resulted from exposure to a toxin. *Id.*

Plaintiffs in toxic tort cases often turn to epidemiology to prove causation. "The field of epidemiology addresses the incidence, distribution and etiology (causation) of disease in human populations by comparing individuals exposed to a particular agent to unexposed individuals to determine whether exposure increases the risk of disease." *In re Silicone Gel Breast Implants Products Liab. Lit.,* 318 F.Supp.2d 879, 892 (C.D. Cal. 2004). Scientists use "relative risk," also referred to as "standard mortality ratio" or "odds ratio," to identify an association between exposure to a chemical and a disease.

> For example, if a study found that 10 out of 1000 women with breast implants were diagnosed with breast cancer and 5 out of 1000 women without implants (the "control" group) were diagnosed with breast cancer, the relative risk of implants is 2.0, or twice as great as the risk of breast cancer without

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> without implants. This is so, because the proportion of women in the implant group with breast cancer is 0.1 (10/1000) and the proportion of women in the non-implant group with breast cancer is 0.05 (5/1000). And 0.1 divided by 0.05 is 2.0.
>
> *Id.*

A relative risk of 1.0 suggests there is no association between the chemical and the disease because the same number of people exposed to the chemical were diagnosed with the disease as those not exposed to the chemical. A relative risk in excess of 1.0, however, demonstrates that those exposed to the chemical are more likely to contract a given disease than those who were not exposed to a chemical. "Where the study properly accounts for potential confounding factors and concludes that exposure to the agent is what increases the probability of contracting the disease, the study has demonstrated *general* causation-that exposure to the agent is capable of causing [the illness at issue] in the general population." *In re Bextra and Celebrex Mktg. Sales Practices and Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1172-73 (N.D. Cal. 2007) (quoting *In re Silicone Gel Breast Implants Products Liab. Lit.,* 318 F.Supp.2d at 893).

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Epidemiology studies are also probative of specific causation. If the relative risk is greater than 2.0, exposure to the chemical more than doubles the risk of developing the disease. That is,

> When the relative risk is 2.0, the alleged cause is responsible for an equal number of cases of the disease as all other background causes present in the control group. Thus, a relative risk of 2.0 implies a 50% probability that the agent at issue was responsible for a particular individual's disease. This means that a relative risk that is greater than 2.0 permits the conclusion that the agent was more likely than not responsible for a particular individual's disease.

Id. (quoting *In re Silicone Gel Breast Implants Products Liab. Lit.,* 318 F.Supp.2d at 893). Consequently, a plaintiff may meet his burden of proving causation by relying on epidemiology studies that establish the relative risk of developing a disease as a result of exposure to a toxin is more than 2.0. *Id.*

In this case, the toxin at issue is benzene and the disease in question is acute myelogenous leukemia. Although ConocoPhillips contends that the true toxin at issue is gasoline, the argument is of little import. There is no real question in the scientific community that exposure to benzene is capable of causing AML. And, whether the toxin is gasoline or benzene, numerous epidemiological studies establish a

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greater than 2.0 relative risk that exposure causes AML. Consequently, there can be little doubt that sufficient scientific evidence supports the causation opinions of Plaintiffs' expert witnesses.

B. General Causation

1. Benzene is A Known Cause of Acute Myelogenous Leukemia.

The need for ConocoPhillips to argue that Plaintiffs must prove that gasoline causes AML is evident from the overwhelming evidence establishing that the benzene component of gasoline is uniquely carcinogenic. As the report from Dr. Peter Infante establishes, benzene has been known to be a powerful bone marrow toxin for more than 100 years.[2] Beginning in the 1920s, cases of benzene-induced leukemia were continually reported in the scientific literature, the first reported case of benzene-related acute leukemia appeared in 1928.[3] During the

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[2] Winslow, C.E.A., Dr., PH, Summary of the National Safety Council Study of Benzol Poising; Journal of Industrial Hygiene (1927).

[3] Expert Report of Peter F. Infante, November 29, 2007 ("Infante First Report"), at 4.

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period from 1930 through the 1970s, hundreds of cases of benzene-induced leukemia were reported in the published scientific literature.[4]

In 1977, Plaintiffs' expert witness, Dr. Peter Infante, conducted the first cohort study of benzene exposed workers while working for NIOSH. In this study, Dr. Infante collaborated with other scientists to analyze the incidence of leukemia among workers manufacturing a rubberized food wrap known as Pliofilm. The results of this study showed that workers exposed to benzene were more than five times likely to contract leukemia than the general population.[5] Follow-up studies performed on the same cohort of individuals have also demonstrated a significant increase in the risk of death from leukemia among those exposed to benzene.[6]

Since the initial publication of the Pliofilm study, numerous other studies have confirmed that exposure to benzene significantly increases the risk of contracting leukemia, specifically AML. For example, in 1978

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[4] Infante First Report at 4-5.

[5] Infante PF, et al. (1977) Leukemia in benzene workers. *Lancet*, ii: 76-78.

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a study of 594 Dow Chemical workers found that workers exposed to benzene were 3.75 times more likely to contract myelogenous leukemia than non-exposed workers.[7] Similarly, a later study regarding the same group of workers demonstrated that those workers exposed to benzene had a 4-fold increased risk of contracting myelogenous leukemia.[8] Other studies have also shown that exposure to benzene substantially increases the risk of developing AML.

To date, the largest epidemiological studies assessing the risk of benzene-induced leukemia have involved exposed workers in China, which have been conducted over the last 15 years. In these studies, researchers from the U.S. National Cancer Institute and from the Institute of Occupational Medicine in China have evaluated the incidence of disease among more than 74,000 workers who were occupationally exposed to benzene and compared the incidence of leukemia to more

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[6] *See, e.g.*, Rinsky RA, et al. (2002) Benzene exposure and hematopoictic mortality: A long-term epidemiologic risk assessment. Am. J. Indust. Med. 42, 474-480 (showing a standard mortality rate of 2.56 for exposed workers).

[7] Ott GM, et al. (1978) Mortality among Individuals Occupationally Exposed to Benzene. Arch Env Health 33, 3-10.

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leukemia to more than 35,000 workers who had no occupational exposure to benzene. The results of these massive studies demonstrated 3.1 relative risk of contracting AML among benzene-exposed workers.[9]

It is not surprising, therefore, that as early as 1977, OSHA declared that there is "conclusive evidence that benzene is a leukemia causing agent." *See* BEN 112 at 170376, 170377. By 1987, OSHA believed that that the link between benzene exposure and leukemia could no longer be "seriously challenged." BEN 227 at 85035. The evidence that benzene causes leukemia is so strong that it is one of a limited number of substances classified by the National Toxicology Program of the Department of Health and Human Services as a "known human carcinogen," similar to tobacco smoke and asbestos. Given the overwhelming evidence, even ConocoPhillips's corporate representative admits that benzene causes AML. *See* Deposition of Jennifer Galvin at 234.

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[8] Bond GG, et al. (1986) An update of mortality among workers exposed to benzene. Br J Indust Med 43, 685-691.

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2. This is a Benzene Exposure Case.

The significance of ConocoPhillips' acknowledgement that benzene is a known cause of AML cannot be overstated. There is also no question that the ConocoPhillips gasoline Mr. Henricksen loaded into his tanker truck for eight years contained benzene. Again, ConocoPhillips' corporate representative admitted that its gasoline contains benzene. *See* Deposition of Jennifer Galvin at 168. According to Ms. Galvin, ConocoPhillips's gasoline contained 1-2% benzene. *Id.* Additionally, ConocoPhillips's MSDS sheets indicate that ConocoPhillips's gasoline contains between 0.1 and 4.9% benzene. *Id.* at 169-70; *see, e.g.,* CON 5004; CON 5005.

Consequently, ConocoPhillips' admission that benzene causes AML is a concession that a significant constituent of its product is capable of causing the exact disease Mr. Henricksen contracted. In other words, despite its contorted efforts to avoid the obvious, ConocoPhillips has conceded general causation. There is no rational

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[9] Yin S-N, et al. (1996) An Expanded Cohort Study of Cancer Among Benzene-exposed Workers in China. Env Health Perpspect, Vol. 4, Supp 6.

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dispute that the benzene in ConocoPhillips' gasoline is capable of causing Mr. Henricksen's AML.

Faced with indisputable evidence of the conspicuous danger that its product poses, ConocoPhillips engages in strained effort to sidestep liability for the damage it has caused by arguing that Plaintiffs not only bear the burden of proving that benzene causes AML, but must also prove that the particular source of benzene is capable of causing AML. According to ConocoPhillips, Plaintiffs' experts cannot opine that benzene in gasoline causes AML unless they first prove that gasoline causes AML. This argument is nothing more than the perennial "ever narrowing box defense:" no matter how strong the evidence is that a toxin causes disease, ConocoPhillips will argue that the evidence is unreliable because it does not match the specific minutia of the present case.

3. The Court Should Not Engage in a Factual Determination as to Which Party's Experts are Correct.

The first fundamental flaw in ConocoPhillips' argument is that a precise match between the circumstance of a case and the body of scientific evidence is not necessary for an expert's opinion to be

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admissible. Regardless of the source of benzene, the scientific literature establishes that benzene causes AML. Even if ConocoPhillips were correct that there is no literature establishing that benzene from gasoline causes AML, it is perfectly reasonable (and reliable) for Plaintiffs' expert to infer that benzene from gasoline is also capable of causing AML when there is no question that benzene from various sources causes the same disease.

The true goal of ConocoPhillips's argument is not to preclude the admission of junk science before the jury, but to obtain a fact finding from the Court that the benzene in its gasoline did not cause Mr. Henricksen's disease. At best, ConocoPhillips' argument that benzene from gasoline is somehow different from benzene from every other source of benzene regulate by OSHA is a potential basis for cross-examining Plaintiffs' experts; it does not establish that the opinion of Plaintiffs' experts that benzene from whatever source is capable of causing AML is so wildly inaccurate that a jury cannot even consider it.

In short, ConocoPhillips asks the Court to utilize the *Daubert* analysis as a mechanism for choosing between the competing theories regarding causation and make a factual determination as to which is

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correct. But, this is exactly the type of flawed *Daubert* analysis that the Supreme Court and the Ninth Circuit have expressly prohibited. Even if there was some debate in the scientific evidence regarding whether benzene causes AML, "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof" remain the "appropriate means of attacking shaky but admissible evidence." *Daubert I*, 509 U.S. at 596, 113 S. Ct. 2786. "If two contradictory expert witnesses [can offer testimony that is reliable and helpful], both are admissible, and it is the function of the finder of fact, not the trial court, to determine which is the more trustworthy and credible." *Dorn*, 397 F.3d at 1195. Consequently, the Court should admit expert testimony that falls within the range of where reasonable experts may differ and permit the jury to decide among the conflicting views. *S.M.*, 262 F.3d at 921. Otherwise, the Court would invade the constitutionally specified role of the jury in resolving factual disputes. *See* Stephen Breyer, *Introduction* to REFERENCE MANUAL ON SCIENTIFIC EVIDENCE at 4 (2nd ed. 2000).

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4. Substantial Evidence Establishes that Benzene from Gasoline Causes AML.

The second flaw in ConocoPhillips's argument is that it is simply wrong in its assertion that there is no scientifically reliable evidence showing that benzene in gasoline is capable of causing AML. Of particular importance is the study conducted by the Pennsylvania Department of Health ("PADOH") in 2000 of the cancer incidence in a community residing at the site of a gasoline spill where approximately 50,000 gallons of gasoline leaked from underground storage tanks, migrating underground and exposing local residents to benzene vapors.[10] In the most recent update of the study, cancer incidence among the members of the gasoline exposed community showed that these individuals were 5.56 times more likely to contract AML than an unexposed population.[11]

ConocoPhillips finds multiple faults in the results reported in this study. For one, ConocoPhillips contends that the risk of leukemia found

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[10] Pennsylvania Department of Health, Bureau of Epidemiology. (December 2001) Tranguch Gasoline Spill Report, Hazelton, Pennsylvania ("PADOH 2001"); Pennsylvania Department of Health, Bureau of Epidemiology. (December 2003) Tranguch Cancer Incidence Study, Updated Through 2002 ("PADOH 2003").

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in this study is "not clear." This, however, is simply inaccurate. The 556% increased risk of AML found in the community was statistically significant at a 95% confidence interval. ConocoPhillips also cites a study conducted by Patel et al.[12] in 2004 to suggest that the data does not show a doubling of the risk of AML among the exposed population. What ConocoPhillips neglects to mention, though, is that the study conducted by the PADOH followed the residences for seven years more than the Patel 2004 study and identified an additional AML, which was statically significant and elevated the relative risk of AML to a 5.6-fold increase.[13]

ConocoPhillips's quibbles with the results demonstrated in the PADOH study is further belied by the multiple other studies that confirm that benzene from gasoline causes leukemia, specifically AML. For example, in 2005 a study was published by Terry et al. regarding the

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[11] PADOH 2003.

[12] Patel AS, et al. (2004) Risk of cancer as a result of community exposure to gasoline vapors. Arch Environ Health, 59: 497-503 ("Patel 2004").

[13] Infante First Report at 22; Compare Patel 2004 with PADOH 2003.

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incidence of leukemia among gas station attendants.[14] The study involved 811 cases of adult leukemia and 637 controls. The results of this study showed that gasoline station attendants employed more than 1 year had an 80% increased risk of contracting AML. Even more significantly, the data showed that employees in the "petroleum industry or manufacturing petroleum products" who were employed more than 1 year had a 620% greater likelihood of contracting AML, indicating a significantly elevated risk of AML among those exposed to petroleum products.[15]

Multiple additional studies also demonstrate that a large body of scientific literature supports the proposition that benzene in gasoline is capable of causing AML. In the 1993 study performed by Jakobsson et al. on the incidence of cancer in Sweden, gas station attendants HAD a much higher chance of contracting AML than do other members of the

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[14] Terry PD, et al. (2005) Occupation, hobbies, and acute leukemia in adults. Leukemia Res 29, 1117-1130 ("Terry 2005").

[15] Terry 2005.

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population.[16] This study showed that male petrol station attendants were 3.6 times more likely to contract AML. In 1990, analogous results were found by Flodin et al.[17] In this case-control study, 86 cases of AML were compared 172 control individuals. Those with occupational exposure to gasoline showed a 2.7-fold increased risk of developing AML.

Other studies further demonstrate that benzene in gasoline causes AML. For one, Shu 1988[18] demonstrated a significant association between maternal occupational exposure to gasoline during pregnancy and an elevated risk of AML among their children. Additionally, Steffan et al. 2004[19] demonstrates a 7.7-fold increased risk of AML among children who lived near a repair garage or a petrol station.

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[16] Jakobsson et al. (1993) Acute Myeloid Leukemia Among Petrol Station Attendants. Arch Environ Health 48, 255-259 ("Jakobsson 1993").

[17] Flodin, U et al. (1990) Acute myeloid leukemia and background radiation in an expanded case-referent study. Arch Env Health 45, 364-366 ("Flodin 1990").

[18] Shu XO, et al. (1988) A population-based case-control study of childhood leukemia in Shanghai. Cancer 62, 635-643.

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ConocoPhillips' excessive concern for inconsequential details and willingness to misrepresent the findings in scientific literature are further demonstrated by its criticisms of these additional studies reflecting that benzene in gasoline causes AML. ConocoPhillips first criticizes the Jakobsson 1993 study by arguing that Jakobsson used flawed methodology as expressed in a letter to the editor by Swane in 1996. However, it fails to inform the Court that in the same issue of the journal, Jakobsson et al. rebutted each and every criticism leveled by Swane and again concluded that "our interpretation of the data does not go further than our findings: benzene exposure from petrol increased the risk of AML for petrol station attendants."[20]

ConocoPhillips also criticizes Terry 2005 because the data does not show a doubling of the risk for AML among gas station attendants. However, ConocoPhillips cannot deny that Terry demonstrates a significantly elevate 80% increase in the risk of AML and also a dose-response relationship between exposure to benzene from gasoline and

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[19] Steffan C et al. (2004) Acute childhood leukemia and environmental exposure to potential sources of benzene and other hydrocarbons; a case-control study. Occup Environ Med 61, 773-778.

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AML. As the data demonstrate, the odds ratio for AML among gas station attendants with less than one year of employment was 1.4 while the odds ratio for employees with more than one year of employment was 1.8. Additionally, it is undeniable that Terry 2005 showed that employees in the "petroleum industry or manufacturing petroleum products" who were employed more than 1 year had a 620% greater likelihood of contracting AML, indicating a significantly elevated risk of AML among those exposed to petroleum products.[21]

ConocoPhillips' criticisms of the studies produced by Plaintiffs' experts are equally unpersuasive. ConocoPhillips faults the Shu study because it is not a study involving occupational exposure and does not account for exposures *not related to leukemia*; however, the substance of these criticisms is unclear. The import of the Shu 1988 study is that the small doses of benzene from gasoline that were passed through the placenta to a fetus were sufficient to cause AML. Additionally, it is a matter of common sense that accounting for exposures that do not

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[20] Jakobsson, Robert et al., Letter to the Editor, (1996) ("Jakobsson 1996").

[21] Terry 2005.

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cause AML will not affect the results of a study attempting to determine what does cause AML.

ConocoPhillips also criticize the findings in Steffan 2004 because the author notes that the possibility of "recall bias" existed in questioning the participants about whether they lived near repair garages or petrol stations. What ConocoPhillips fails to mention is that the authors specifically addressed this concern. In noting that the presence of a neighboring garage or petrol station near a child's home is "quite unambiguous," the authors determined that recall bias should have a "limited over or under declaration."

Finally, ConocoPhillips criticizes the Flodin 1990 study because the confidence interval for the reported result was only 90%, instead of the preferred 95%. This effectively means if the study were repeated, the same results should be reached 90% of the time. In other words, the odds that the results were reached merely by chance were just 10%. While there is no doubt that increased certainty is preferred, this slight variance in certainty has little consequence in a legal proceeding in which the burden of proof is simply "more likely than not." In fact, it is remarkable that ConocoPhillips could suggest that the Court should

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wholly disregard the results of this study when it is clear that the scientific community thought the study of sufficient merit to publish it in a peer reviewed journal. The purpose of the *Daubert* analysis is to exclude evidence that is not founded in good science, not to exclude evidence that the scientific community relies on in the everyday practice of the relevant field.

The opinion that benzene in gasoline is capable of causing leukemia is further supported by evidence showing significant genotoxic effects as a result of very low exposure to benzene from gasoline vapor. Significant elevations in damage to the DNA in leukocytes (white blood cells) has been shown to result from exposure to benzene in gasoline.[22] This is important because leukemia is a cancer of the blood or bone marrow that this characterized by an abnormal proliferation of blood cells, usually white blood cells. In particular, AML is a cancer of the myeloid line of white blood cells. DNA damage in these white blood cells

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[22] *See, e.g.*, Hogstedt B, et al. (1991) Gasoline pump mechanics had increased frequencies and sizes of micronuclei in lymphocytes stimulated by pokeweed mitogen. Mutat Res 263, 51-55 ("Hogstedt 1991"); Oesch F, et al. (1995) DNA single strand break analysis in mononuclear blood cells of petrol pump attendants. Ind Arch Occup Environ Health 67, 35-39 ("Oesch 1995"); Santos-Mello R. (1992) Cytogenetic studies on gas station attendants. Muat Res 280, 285-290 ("Santos-Mello 1992").

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cells has been demonstrated at benzene exposures as low as 0.1 ppm.[23] Of particular importance, multiple studies have shown DNA damage among petrol pump attendants exposed to gasoline,[24] leading one group of scientists to conclude that the significant increase in DNA damage among gasoline pump mechanics is "probably caused by the benzene content of the gasoline."[25] These adverse effects on DNA from exposure to benzene in gasoline are consistent with the results of animal studies showing chromosomal damage in mice at exposures as low as 40 ppb and 100 ppb.[26]

At best, ConocoPhillips' nitpicking is a matter for cross-examination. Again, the only issue before the Court is whether the opinions of Plaintiffs' expert witnesses are sufficiently reliable to admit to a jury. As the Ninth Circuit has recognized, one significant indicia of

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[23] Nilsson RI, et al. (1996) Genotoxic effects in workers exposed to low levels of benzene from gasoline. Am J Ind Med 30, 317-324.

[24] Hogstedt 1991; Oesch 1995; Santo-Melo 1992.

[25] Hogstedt 1991.

[26] Au WW, et al. (1991) Chromosome aberrations in lymphocytes of mice after sub-acute low-level inhalation exposure to benzene. Mutat Res 260, 219-224; Ward JB, et al. (1992) The

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reliability is whether expert's opinion arose "naturally and directly out of research [the expert] conducted independent of the litigation." *Daubert II*, 43 F.3d 1317. Here, Dr. Infante reached the conclusion that benzene from gasoline can cause AML long before this litigation began. In 1993, Dr. Infante and Dr. Phillip Enterline were asked by the American Petroleum Institute to review three recent studies[27] regarding the risk of leukemia among gasoline exposed workers as part of the International Symposium on the Health Effects of Gasoline in 1991. In his review, Dr. Infante noted that the studies showed an elevated risk of leukemia among gasoline-exposed workers and concluded that this elevated risk is "probably related to the benzene content of gasoline."[28] Similarly, Dr. Enterline noted that all three of the studies reviewed showed an excess of leukemia "particularly acute myelogenous leukemia" among gasoline

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mutagenic effects of low level sub-acute inhalation exposure to benzene in CD-1 mice. Mutat Res 268, 49-57.

[27] Wong O, et al. (1993) Health Effects of Gasoline Exposure: II. Mortality Patterns of Distribution Workers in the United States. Environ Health Perspect 101, 63-76; Schnatter AR, et al. (1993) A Retrospective Mortality Study among Canadian Petroleum Marketing and Distribution Workers. Env Health Perspec 101, 85-99; Ruston L. (1993) A 39-Year Follow-up of the U.K. Oil Refinery and Distribution Center Studies: Results for Kidney Cancer and Leukemia. Environ Health Perspect 101, 77-84.

[28] Infante PF. (1993) State of the science on the carcinogenicity of gasoline with particular reference to cohort mortality study results. Environ Health Perspect 101, 105-109.

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distribution workers and concluded that "there is evidence of a relationship between gasoline exposure and acute myelogenous leukemia and it is possible that this is due to the benzene content of gasoline."[29] The reliability of these opinions is especially strong given that these opinions are contrary to the interests of the organization for which the opinions were given.

Even though the majority of the scientific literature cited above occurred after the most recent Monograph published by the International Agency for Research on Cancer ("IARC"), IARC concluded in 1989 that gasoline is a "possible carcinogen to humans." Since that time, the wealth of information cited above has only strengthened the causal association between benzene in gasoline and AML. Given the recent studies showing a strong correlation between AML and exposure to benzene in gasoline (well in excess of a doubled risk), and the data establishing the genotoxic effect of low exposures to benzene from gasoline vapor, it is absurd for ConocoPhillips to argue that there is no

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[29] Enterline PE. (1993) Review of new evidence regarding the relationship of gasoline exposure to kidney cancer and leukemia. Environ Health Perspect 101, 101-103.

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reliable basis underlying the opinion of Plaintiffs'' experts that benzene from gasoline is capable of causing AML.

C. Specific Causation

The evidence relied on by Plaintiffs' experts in their opinion that Mr. Henricksen's AML was caused by his exposure to benzene from ConocoPhillips' gasoline is equally substantial. It is well established in the scientific literature that a doubling of the risk for AML occurs at very low doses of benzene. In fact, because benzene in a mutagenic carcinogen, there is no known level at which AML does not occur. Nevertheless, the dose reconstruction analysis performed by Dr. Sawyer and Mr. Kaltofen establish that Mr. Henricksen's exposure was far in excess of any minimum level needed to show that his AML was caused by exposure to benzene from ConocoPhillips' product.

1. A Dose Calculation is Not Necessary for A Cancer-Causing Mutagen Such as Benzene.

Throughout its briefing, ConocoPhillips repeatedly asserts the general tenet of toxicology that "the dose makes the poison," citing Bernard D. Goldstein and Mary Sue Henifin, *Reference Guide on Toxicology*, *in* REFERENCE MANUEL ON SCIENTIFIC EVIDENCE 403 (2nd ed.

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2000). There is no question that this proposition is generally true. However, ConocoPhillips' selective citation to this article is misleading.

As with any general proposition, there are exceptions to the tenant that "the does makes the poison." In fact, the same article cited by ConocoPhillips also provides that:

> Certain genetic mutations, such as those leading to cancer . . ., are believed to occur without any threshold. In theory, the cancer-causing mutation to the genetic material of the cell can be produced by any one molecule of certain chemicals. The no threshold model led to the development of the one hit theory of cancer risk, in which each molecule of a cancer-causing chemical has some finite possibility of producing the mutation that leads to cancer. This risk is very small, since it is unlikely that any one molecule of a potentially cancer-causing agent will reach that one particular spot in a specific cell and result in the change that then eludes the body's defenses and leads to a clinical case of cancer. However, the risk is not zero.

Id. at 407-08. Consequently, while causation in a toxic tort case typically requires evidence of exposure to a level of the toxin capable of causing harm, the analysis is inapplicable to "substances that exert toxicity by causing mutations leading to cancer." *Id.* at 426.

This is exactly the position that OSHA has taken in its regulation of workplace carcinogens such as benzene, which has been upheld by the

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D.C. Circuit Court. *See* 29 C.F.R. § 1990.143(h) ("No determination will be made that a "threshold" or "no-effect" level of exposure can be established for a human population exposed to carcinogens in general, or to any specific substance"); *Public Citizen Health Research Group v. Tyson*, 796 F.2d 1479, 1498 (D.C. Cir. 1986). As OSHA noted in 1977, the best available scientific evidence indicates that no safe level for exposure to a carcinogen, including benzene, can be established to exist." CON 5032 at 22517. OSHA's position is based, in part, on a statement from NIOSH that "It is not possible at the present time to establish an exposure level at which benzene may be regarded to be without danger." *Id.* at 22521.

Even OSHA's current required limits for benzene exposure in the workplace recognize that lack of any threshold at which benzene does not cause leukemia. Since 1987, OSHA has limited benzene exposure to 1 ppm on an 8-hour time-weighted average and 5 ppm for short-term exposure limit. BEN 227 at 34460. OSHA set this limit because it was "the lowest feasible level for industry in general," and encouraged employers to achieve exposures below 0.5 ppm if possible. *Id.* at 34461. However, even at this level, OSHA recognizes that there will be excess

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excess deaths from leukemia resulting from benzene exposure. According to OSHA's estimation, there will be "95 excess leukemia deaths per 1000 exposed employees *for working lifetime* of exposure to 10 ppm benzene and *10 excess deaths per 1000 at 1 ppm*." *Id.* at 34463 (emphasis added). This translates into a relative risk of contracting leukemia at 2.43 even at OSHA's current required benzene exposure limit.[30]

In short, the best scientific evidence available establishes that there is no safe level of exposure to benzene. Instead, as with other mutagenic carcinogens, the cancer-causing mutations can occur at any level. Consequently, the general maxim that "the dose makes the poison" is simply inapplicable in determining whether benzene caused Mr. Henricksen's AML.

2. Benzene Causes AML at Extremely Low Doses and Low Levels

Even if there is some no-effect level of exposure to benzene, Dr. Infante's report establishes that extremely low exposures to benzene more than double the risk of contracting AML. According to Dr. Infante,

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[30] Infante First Report at 8.

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a significantly elevated risk of AML exists at cumulative exposures[31] to benzene as low as 1.5 ppm-years and 6.7 ppm-years and significantly increased risk of contracting leukemia exists at average exposure levels[32] as low as 0.2 ppm and 0.8 ppm. Dr. Infante's opinion is well supported by epidemiological studies.

In Hayes 1997, the investigators analyzed the dose-response resulting from an enormous cohort of more than 74,000 Chinese workers who were exposed to benzene. In this study, the scientists observed that those exposed to average benzene levels of less than 10 ppm showed a 3.2-fold increased risk of contracting AML.[33] Although the study does not explicitly state the cumulative dose at which these results showed a 3.2 relative risk, the authors reported to the State of California

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[31] The cumulative exposure figure of ppm-years is calculated by multiplying the 8-hour time-weighted average exposure experienced by a person by the number of years of exposure.

[32] The average exposure figure is calculated simply by averaging a person's exposure over time.

[33] Hayes RB, et al. (1997) Benzene and the dose-related incidence of hematologic neoplasms in China. J Natl Cancer Inst 89, 1065-1071 ("Hayes 1997").

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California that this risk occurred at just 6.7 ppm-years.[34]

Numerous other studies also establish a greater than doubling of the risk of AML at very low levels of benzene exposure. For instance, in 2001, Gray et al. published a case-control study on petroleum industry workers and found a 7-fold increased risk of contracting leukemia at exposure intensities as low as 0.8 – 1.56 ppm.[35] The authors also evaluated the data for leukemia subtypes. For AML, exposure to a maximum intensity level of just 0.6 ppm to 4.8 ppm revealed a 5.71 odds ratio for AML. Even when the data for this range of exposure was compared to the data from individuals exposed to benzene but less than 0.6 ppm, the odds ratio for AML was 4.79.[36] This data shows a readily apparent association between low intensity exposures to benzene and AML.[37] Finally, the analysis of cumulative benzene exposure also showed a very high relative risk for leukemia at very low levels of

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[34] *See* California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. (2001) Public Health Goal for Benzene in Drinking Water (citing Hayes 1997).

[35] Gray C, et al. (2001) Lympho-haematopoictic Cancer and Exposure to Benzene in the Australian Petroleum Industry. Monash University and Deakin University ("Gray 2001").

[36] Id.

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exposure. At just 1.44 ppm-years, workers showed a 4.1 relative risk for leukemia, and at 2.78 ppm-years, workers showed a 4.7 relative risk for leukemia.[38]

Glass et al. also conducted a series of case-control studies on the same population of Australian refinery workers, which also demonstrated an increased risk of leukemia from low cumulative exposures to benzene. In 2003, Glass et al. reported that cumulative exposures of more than 8 ppm-years resulted in a more than 7-fold increase in leukemia. The authors also noted that the data established that the "risk of leukemia was increased at cumulative exposures above 2 ppm-years and with intensity of exposure of the highest exposed job over 0.8 ppm." From these data, the authors concluded that "no evidence was found of a threshold cumulative exposure below which there was no risk."[39]

The group of studies arising from PADOH investigation of the large

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[37] Infante First Report at 10.

[38] Gray 2001.

[39] Glass DC, et al. (2003) Leukemia risk associated with low-level benzene exposure. Epid 14, 569-577.

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large gasoline spill in Pennsylvania also provided compelling, and particularly relevant, evidence that very low doses of benzene cause AML. As one paper noted, the Agency for Toxic Substances and Disease Registry ("ASTDR") estimate the cumulative benzene exposure for the residents of the homes at issue to be just 0.03 ppm-years over 3 years.[40] The highest cumulative dose experienced by these residents was 2 ppm-years. These estimates were generated by data collected by the PADOH prior to remediation of the site. Despite this exceedingly low exposure to benzene from gasoline, Patel 2004 observed a 4.4 relative risk of leukemia, and the PADOH, which followed the community for 7 years longer than Patel et al., noted a 5.56 relative risk for AML. From this information Patel et al. concluded that their study provided support for the growing body of evidence that low-level benzene exposure causes leukemia.

3. Neil Henricksen Was Exposed to Substantial Amounts of Benzene From ConocoPhillips's Gasoline.

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[40] Patel 2004.

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Given the extraordinarily low level of benzene required to double the risk of contracting AML, there can be little doubt that the dose suffered by Mr. Henricksen was more than sufficient to double his risk of contracting AML. As Dr. Sawyer and Mr. Kaltofen demonstrate, Mr. Henricksen suffered a cumulative dose of benzene in excess of 8 ppm-years. Although each doctor utilized a different methodology and relied on different studies, the results reached by Dr. Sawyer and Mr. Kaltofen are remarkably similar.

i. Dr. Sawyer Accurately Estimated Mr. Henricksen's Cumulative Benzene Exposure.

First, ConocoPhillips' criticisms of Dr. Sawyer's dose calculation are extraordinarily disingenuous. To calculate Mr. Henricksen's benzene exposure, Dr. Sawyer first determined the amount of time Mr. Henricksen spent loading his truck with gasoline. Relying on his interview with Mr. Henricksen, Dr. Sawyer determined that Mr. Henricksen top-loaded his truck 25 to 30 times per week and each event took 30 to 45 minutes, "sometimes longer." Taking the means of these two ranges, Dr. Sawyer determined that Mr. Henricksen spent 1031.2 minutes each week loading his tanker truck with fuel (i.e. 27.5 loading events lasting approximately 37.5 minutes). This is the equivalent of

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17.2 hours per week, which is 43% of a 40-hour work week, or 29.9% of Mr. Henricksen's usual 55 to 60 hour work week.

Although Mr. Henricksen testified that he worked 55-60 hours a week, Dr. Sawyer determined the percentage of time spent loading his truck based on a 40-hour work week because cumulative dose estimates are made by averaging the dose to a toxin over a standard 40-hour work week, which is the generally accepted benchmark in time weighted average calculations. By making this estimate, Dr. Sawyer assumes that Mr. Henricksen had no exposure to benzene other than what occurred during the loading of his vehicle, which substantially underestimates his actual exposure. Dr. Sawyer does not account for the benzene exposure Mr. Henricksen would have received while unloading his truck, from the fumes created by occasional spills, or from dermal absorption of benzene that occurred when Mr. Henricksen came in contact with ConocoPhillips' gasoline. Consequently, despite ConocoPhillips' objections, Dr. Sawyer's dose estimate is extremely conservative.

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To determine the amount of exposure Mr. Henricksen received for the 43% of a 40-hour work week he spent loading his truck with gasoline, Dr. Sawyer turned to peer-reviewed published literature to obtain an estimate of the benzene exposure that occurs during the top loading of a gasoline tanker without any vapor recovery system in a closed terminal. For this data, Dr. Sawyer turned to the results published in 1999 by Kawai et al.[41] In this study, the authors measured the mean benzene exposure levels during the loading of a tanker truck with gasoline, which the authors found to be 5.2 ppm. Dr. Sawyer chose this study because, like Mr. Henricksen, it involved the top-loading of gasoline without vapor recovery.

Moreover, the benzene content of the gasoline studied in Kawai 1999 ranged from 1.15% to 1.62% across the different brands and 0.91% to 2.08% by location. This benzene content is very similar to the benzene content in ConocoPhillips' gasoline. Moreover, ConocoPhillips' corporate representative concedes that ConocoPhillips' gasoline contains 1-2% benzene. *See* Deposition of Jennifer Galvin at 168. This

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[41] *See* Kawai T, et al. (1991) Exposure to vapors of benzene and other aromatic solvents in

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is also consistent with ConocoPhillips' MSDS forms, which indicate that ConocoPhillips gasoline contains between 1 and 4.9% benzene. CON 5004, CON 5005. In its motion to strike Dr. Sawyer's testimony ConocoPhillips faults Dr. Sawyer for relying on this study because it believes that the gas transported by Mr. Henricksen contained only 1% benzene. However, given the evidence above, ConocoPhillips cannot reasonably deny that Dr. Sawyer has a valid basis for determining that the benzene content of the gasoline measured in Kawai 1999 was reasonably similar to the benzene content of ConocoPhillips' gasoline.[42]

From this data, Dr. Sawyer was able to estimate the cumulative benzene dose Mr. Henricksen received while loading ConocoPhillips gasoline in his tanker truck. Mr. Henricksen was exposed to 5.2 ppm of benzene for 43% of a 40-hour work week, which yields an 8-hour time

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tank truck loading and delivery. Bull. Envirn. Contam. Toxicology 46, 1-8 ("Kawai 1999").

[42] Oddly, ConocoPhillips also criticizes Dr. Sawyer for assuming that ConocoPhillips' gasoline contains 2% benzene. This objection is founded on an assumption made in Dr. Sawyer's report and restated in his deposition. However, this assumption had absolutely no bearing on Dr. Sawyer's dose calculation. The benzene content assumed in Dr. Sawyer's calculation is the content reflected in Kawai 1999, which ranged from 1.15% to 1.62% across the different brands and 0.91% to 2.08% by location. This content is almost precisely the 1-2% benzene content that ConocoPhillips' corporate representative testified was in ConocoPhillips' gasoline. *See* Deposition of Jennifer Galvin

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weighted average of 2.24 ppm TWA (i.e. 5.2 ppm x .43 = 2.24). However, because Mr. Henricksen actually delivered diesel fuel 50% of the time, which does not result in as significant benzene exposures as gasoline, his actual exposure over a 40-hour work week was 1.12 ppm TWA (i.e. ½ of 2.24 ppm TWA). Because Mr. Henricksen delivered ConocoPhillips gasoline for 8 years, his cumulative dose to benzene from this activity was 8.9 ppm-years (i.e. 1.12 ppm TWA x 8 years).

ConocoPhillips does not fault the method of calculations used by Dr. Sawyer in reaching this estimate. Instead, ConocoPhillips attempts to discredit Dr. Sawyer's dose calculation by attacking the data he relied on in making the calculations. First, ConocoPhillips argues that Dr. Sawyer erred in his estimate of the amount of time Mr. Henricksen spent loading his truck. ConocoPhillips contends that Mr. Henricksen loaded his truck, at most 21 times per week, not 27.5 times per week. ConocoPhillips is simply wrong. The testimony from Mr. Henricksen cited by ConocoPhillips actually provides as follows:

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at 168. It is hard to imagine how Dr. Sawyer could have found a more accurate study to rely on in making his calculations in this case.

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Q: And during a given week working of Husky, how many different offload, offloading events on average would you have?

A: It was just, for Husky, it's hard to say, two to five, you know.

Q: Two to five per what?

A: That's how many loads I would haul. So that's how many unloads I would do a day.

Q: Two to five per day?

A: Yeah.

Q: How many days per week?

A: I pretty much worked 5 ½, 6 days a week.

Q: So if we do the math, that gets us somewhere near like 25 to 30 offloading events per week, do you agree with that or not agree with that?

A: Yeah, that would be - -

Q: Is that about right?

A: Yes.[43]

Instead, of demonstrating that Dr. Sawyer's factual assumption were wrong, ConocoPhillips establishes that Dr. Sawyer accurately estimated

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[43] *See* Exhibit B to ConocoPhillips' Motion to Exclude the Testimony of Plaintiff's Expert William Sawyer at 193-94. ConocoPhillips also cites the Court to p. 231, l.1-6, but this exchange merely confirms Mr. Henricksen's prior estimate that he delivered between 2 and 5 loads a day while he was delivering ConocoPhillips' gasoline.

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that Mr. Henricksen delivered between 25 and 30 loads a week, the mean of which is 27.5.

ConocoPhillips also criticizes Dr. Sawyer for relying on the exposure data for the short-term tasked based samples reported in Kawai 1999, suggesting that Dr. Sawyer testified that his reliance on this data was erroneous. Again, the actual testimony cited by ConocoPhillips establishes that ConocoPhillips' complaint is specious. The first reference to Dr. Sawyer's deposition simply establishes that Dr. Sawyer relied on the measurements of benzene exposure during top-loading and multiplied that number by 43% to obtain a time weighted average exposure for Mr. Henricksen.[44]

In the second deposition reference cited by ConocoPhillips, Dr. Sawyer simply states that it would be erroneous to calculate a time weighted average based on the task based measurement "unless the only exposure the individual had" was during that task.[45] What ConocoPhillips fails to recognize is that the assumption Dr. Sawyer

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[44] *See* Exhibit A to ConocoPhillips' Motion to Exclude the Testimony of Plaintiff's Expert William Sawyer at 174:1-10.

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made in his calculation is precisely that the only exposure Mr. Henricksen suffered was while loading gasoline in his tanker. More importantly, ConocoPhillips apparently fails to appreciate that this assumption *underestimates* Mr. Henricksen's exposure because there is no question that he was also exposed to benzene while unloading the gasoline and when the gasoline spilled on his clothing, which are not accounted for in Dr. Sawyer's calculation. In other words, Dr. Sawyer has not assumed that Mr. Henricksen was exposed to benzene for his entire 55 to 60 hour work week, but calculated a time weighted average based on the fraction of time he loaded gasoline into his truck and assumed that he had no exposure for the remaining 40.3 hours of his work week.

Next, ConocoPhillips faults Dr. Sawyer for failing to subtract Mr. Henricksen's vacation time from his dose calculation. Without any citation, ConocoPhillips claims that Mr. Henricksen took 3 weeks of vacation each year for 8 years and that Dr. Sawyer should have, therefore, assumed that Mr. Henricksen was only exposed for 7 years.

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[45] *See* Exhibit A to ConocoPhillips' Motion to Exclude the Testimony of Plaintiff's Expert

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ConocoPhillips' argument tests the bounds of credibility. For one, even if Mr. Henricksen took a 3-week vacation each year, he worked 7.538 years, not 7 years. If this, number is substituted into Dr. Sawyer calculation, the resulting cumulative exposure is 8.44 ppm-years as opposed to 8.9 ppm-years. Moreover, the body of peer-reviewed studies from which Mr. Henricksen's benzene exposure was calculated do not subtract out vacation time;" instead, simply calculate the a worker's cumulative exposure by multiplying the number of years worked by the 8-hour time weighted average.

ConocoPhillips also contends that Dr. Sawyer's opinions are unreliable because he utilized the arithmetic mean benzene exposure from the Kawai 1999 study as opposed to the geometric mean. Again, ConocoPhillips incorrectly argues that Dr. Sawyer admitted that this was an error. While Dr. Sawyer admitted that using the geometric mean would be a more appropriate method for determining the "central values" experienced by the drivers in Kawai 1999, he did not testify that relying on the arithmetic mean was an error.

William Sawyer at 182:20-25.

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Instead, he noted that the purpose of using a geometric mean is to suppress the effect of high "outlier" values. However, Mr. Henricksen's exposure was, in fact, a high outlier exposure. Unlike most exposures in an open or roofed terminal, Mr. Henricksen's exposure occurred in an unusually enclosed terminal with a roof and partial walls on two sides. *See* Deposition of Neil Henricksen at 178-81; Exhibit 5. Relying on a geometric mean in this case would, therefore, underestimate Mr. Henricksen's true exposure.

Additionally, this argument has little practical significance to Dr. Sawyer's calculation. Even if the geometric mean from Kawaii 1999 were used, Mr. Henricksen's cumulative exposure to benzene was 4.9 ppm-years, which is substantially higher than the dose required to double the risk of contracting AML. With either calculation, ample evidence exists establishing that the benzene from ConocoPhillips' gasoline caused Mr. Henricksen's AML.

Finally, ConocoPhillips faults Dr. Sawyer for relying on the "Canadian agency driver" dose calculations reported in Verma 1998 in determining Mr. Henricksen's benzene dose, as opposed to the lower calculations for terminal drivers. This argument is strange given the

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pages of arguments that Dr. Sawyer erred in relying on the data presented in Kawai 1999 for his calculations. In reality, although Dr. Sawyer cited Verma 1998 in his report, he did not rely on Verma 1998 for his does estimate. This was because Verma's calculations were based on open terminal top-loading, which would have significantly underestimated Mr. Henricksen's actual exposure. To estimate Mr. Henricksen's dose based on the data in Verma would require a conversion factor estimating the difference between benzene levels in top-loading operations in an open terminal and the same operations in a closed terminal, which is exactly what Mr. Kaltofen did in his estimations of Mr. Henricksen's dose. Remarkably, either method of calculating Mr. Henricksen's cumulative benzene dose results in virtually the same result.

In the end, ConocoPhillips' tepid criticisms of Dr. Sawyer's dose calculations are nothing more than cross-examination material. Despite every effort, ConocoPhillips has not shown that Dr. Sawyer's calculations are wrong, or even slightly inaccurate. As demonstrated above, Dr. Sawyer dose estimate is well founded on valid data and unquestioned methodology. Consequently, ConocoPhillips' motion to exclude his

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exclude his testimony must be denied.

ii. Mr. Kaltofen's Cumulative Dose Calculations are Also Accurate and Remarkably Similar to Dr. Sawyer's Calculations.

ConocoPhillips next criticizes the dose calculation of Mr. Kaltofen. Although Mr. Kaltofen used a different method for estimating Mr. Henricksen's cumulative dose, his results are virtually identical to Dr. Sawyer's. As with its other complaints, ConocoPhillips' objections to Mr. Kaltofen's testimony are flimsy and unpersuasive when stripped of their misdirection.

For his dose calculation, Mr. Kaltofen turned to peer-reviewed literature to obtain an initial estimate of Mr. Henricksen's time weighted average exposure while loading ConocoPhillips gasoline. Mr. Kaltofen determined that an appropriate assumed time weighted average for tanker truck operators loading gasoline was 0.38 ppm based on the results of two studies. Of particular significance to Mr. Kaltofen's assumption were the results published in Verma 2004.[46] In this study,

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[46] Verma, DK. (2004) A Simultaneous Job- and Task-Based Exposure Evaluation of Petroleum Tanker Drivers to Benzene and Total Hydrocarbons. J. Occupational and Environ. Hygiene ("Verma 2004").

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tanker truck drivers working shifts longer than 8 hours, experienced time weighted average exposures to benzene between 0.25 ppm and 0.48 ppm.[47] As Mr. Kaltofen testified, his estimate of 0.38 was chosen, in large part, because it fell within this range of potential time weighted averages.

Additionally, Mr. Kaltofen's estimate is supported by the results in Irving & Grumbles 1979.[48] In that study, the authors similarly measured the benzene exposure for top loading a tanker truck without vapor recovery and determined that for an 8-hour work day consisting of 4 tanker loads of fuel, workers were exposed to a 0.38 ppm.[49] This was consistent with the range of time weighted averages found by Verma 2004 and supportive of Mr. Kaltofen's determination of the 0.38 point within that range.

ConocoPhillips' first complaint with regard to Mr. Kaltofen's opinion is that Mr. Kaltofen committed some great gaffe by citing Irving &

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[47] Verma 2004 at 729, 735.

[48] Irving, W.S., et al., "Benzene Exposures During Gasoline Loading at Bulk Marketing Terminals," Am. Indus. Hygiene Assn (1979) ("Irving & Grumbles 1979").

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Grumbles 1979 in his report and relying primarily on Verma 2004 at his deposition. ConocoPhillips' contentions are misguided. At no time did Mr. Kaltofen change his opinion or disavow any reliance on Irving & Grumbles. As he stated in his deposition, Mr. Kaltofen relied primarily on Verma 2004 because the data in that study was adjusted for standard temperature and pressure.[50] Nevertheless, he also relied on the results from Irving & Grumbles 1979 as further support for his determination that 0.38 was an appropriate estimation. Essentially, ConocoPhillips contends that Mr. Kaltofen's opinion in this regard is unreliable because it is supported in multiple peer-reviewed papers.

ConocoPhillips also complains that Mr. Kaltofen erred in relying on the data from Verma 2004. For one, ConocoPhillips alleges that Mr. Kaltofen erred by assuming that the tanker truck drivers in Verma 2004 were top loaders when, in fact, some of the drivers loaded gasoline from the bottom. Apparently, ConocoPhillips fails to recognize that even if this allegation is true it only establishes that Mr. Kaltofen's estimates are

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[49] Irving & Grumbles 1979 at 472.

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conservative because it is well established that bottom-loading creates substantially less benzene exposure than top loading.[51] ConocoPhillips also suggests that Mr. Kaltofen misunderstood Verma 2004 and should have relied on the exposure limits of 0.12 reported in Verma 2004 instead of the time weighted averages. ConocoPhillips' contention is odd given that Verma 2004 stated that the exposure limit should be adjusted for workers such as Mr. Henricksen who worked more than 8 hours per day.[52]

From this 0.38 time weighted average estimate, Mr. Kaltofen is able to calculate Mr. Henricksen's benzene dose based on the other factors in Mr. Henricksen's career. Because the tanker drivers in Verma 2004 loaded gasoline approximately 80% of the time and Mr. Henricksen loaded gasoline approximately 50% of the time, Mr. Kaltofen first adjusted the 0.38 estimate by 5/8 to determine Mr. Henricksen's time weighted average for the actual percentage of time he spent loading

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[50] *See* Exhibit to ConocoPhillips' Motion to Exclude the Testimony of Marco Kaltofen at 40-41.

[51] *See, e.g.,* Irving & Grumbles 1979 at 472; Verma, Dave K., "Hydrocarbon Exposures at Petroleum Bulk terminals and Agencies" at 648, Am. Indus. Hyg. Assn. J. (1992) ("Verma 1992")

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loading gasoline, which resulted in a time-weighted average of 0.2375-ppm. Mr. Kaltofen next multiplied this figure by his estimate that Mr. Henricksen worked for seven years, resulting in a cumulative dose estimate of 1.6625 ppm-years. As with Dr. Sawyer, Mr. Kaltofen's dose calcuations are very conservative because they assume that Mr. Henricksen's only exposure to benzene from ConocoPhillips gasoline was during loading and because he assumed that Mr. Henricksen worked only 7 years instead of the 8 years reflected in his deposition.

ConocoPhillips' primary complaint with respect to the final step in Mr. Kaltofen;s analysis. Because Plaintiffs' experts learned that the data in the Verma 2004 study was based on measurements taken primarily at open facilities with no roof or walls,[53] it drastically underestimated Mr. Henricksen's cumulative exposure because the terminal where he loaded ConocoPhillips' gasoline was roofed and had two partial walls, which substantially reduced the dissipation of the benzene vapors and increased Mr. Henricksen's exposure by hampering the natural

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[52] Verma 2004 at 735.

[53] *See* Deposition of William Sawyer at 23-25.

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ventilation.[54] To estimate this increased exposure, Mr. Kaltofen again turned to peer-reviewed literature to determine a ratio of exposure between open and closed terminals.

In 1987, Swedish scientists determined such a ratio in Nordlinder 1987.[55] In this study, the authors compared the benzene exposure levels and found that benzene levels in "enclosed" terminals were 5 times higher than benzene levels in open terminals.[56] These enclosed terminals where higher benzene levels were observed are described as "buildings which give rise to less effective natural ventilation . . . ."[57] Because the building where Mr. Henricksen loaded ConocoPhillips' gasoline was uniquely "enclosed," Mr. Kaltofen reasonably relied on this data for the open/closed terminal ratio needed to accurately reflect Mr. Henricksen's exposure. In fact, given the unusual amount of enclosure at the ConocoPhillips terminal, this ratio in all likelihood underestimates

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[54] Nordlinder 19

[55] Nordlinder, Rolf et al. "Exposure to Benzene at Different Work Places in Sweden," Annual Occ. Hygiene (1987) ("Nordlinder 1987").

[56] Nordlinder 1987 at 349.

[57] Nordlinder 1987 at 349.

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the true exposures suffered by Mr. Henricksen. Based on this peer-reviewed estimate, Mr. Kaltofen finally determined that Mr. Henricksen's cumulative dose to benzene was approximately 8.3125 ppm-years (i.e. 1.6625 ppm-years x 5).

ConocoPhillips lodges multiple complaints regarding Mr. Kaltofen's reliance on the ratio described in Nordlinder 1987. For one, ConocoPhillips complains that Nordlinder 1987 is not an appropriate study to rely on because the benzene content in Swedish gasoline his higher than the benzene content in ConocoPhillips' gasoline. This, again, is an effort at misdirection. Mr. Kaltofen does not rely on Nordlinder 1987 to establish the amount of Mr. Henricksen's benzene exposure. He relies on Nordlinder 1987 to show the ratio between benzene levels at open and enclosed terminals. Whether the benzene content the gasoline at issue were 1% or 10%, the ratio between open and enclosed terminals would remain the same. The amount of benzene in the gasoline tested by the authors in Nordlinder 1987 would be relevant only if Mr. Kaltofen used the data from that study to estimate the amount of exposure experienced by Mr. Henricksen.

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Finally, ConocoPhillips argues that Mr. Kaltofen failed to account for several minute factors that may have influenced the results of Nordlinder 1987, such the extent of enclosure at the enclosed terminals. However, as with every other complaint leveld by ConocoPhillips, these complaints regarding this level of minute detail do not bear on the admissibility of Mr. Kaltofen's opinion. At best, these types of complaints may provide some material for cross-examination, not the wholesale exclusion of Mr. Kaltofen's testimony. As shown above, Mr. Kaltofen's opinions are well founded in peer-reviewed data and calculated based on unquestioned methodology. Therefore, Mr. Kaltofen's testimony is admissible.

4. Neil Henrickson's Dose of Benzene from ConocoPhillips' Gasoline Was Sufficient to Cause His AML.

Given the overwhelming evidence outlined above, it is not surprising that all four of Plaintiffs' retained expert witnesses and one of Mr. Henricksen's treating physicians have all come to the conclusion that Mr. Henricksen's AML was caused by exposure to benzene from ConocoPhillips' gasoline. As multiple courts have determined, a plaintiff may prove specific causation by relying on epidemiology studies that

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establish a relative risk of developing a disease as a result of exposure to a toxin that is greater than 2.0. *See, e.g., In re Bextra and Celebrex Mktg. Sales Practices and Prod. Liab. Litig.*, 524 F. Supp. 2d 1166, 1172-73 (N.D. Cal. 2007) *In re Silicone Gel Breast Implants Products Liab. Lit.,* 318 F.Supp.2d 879, 8923 (C.D. Cal. 2004). Here, the reports of Drs. Infante and Sawyer list numerous peer-reviewed epidemiological studies showing that exposure to benzene causes AML at very low levels and low doses, whether the source of the benzene is gasoline or some other product. The relative risks found as a result of exposure to benzene published in the peer-reviewed literature include 2.7,[58] 3.2,[59] 3.6,[60] 5.6,[61] and 6.2.[62]

Even though it is not necessary to establish a dose of a genotoxic agent such as benzene to prove that it is a causative factor in an illness so strongly linked with exposure, the peer-reviewed literature shows that

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[58] Flodin 1990.

[59] Hayes 1997.

[60] Jakobsson 1993.

[61] PADOH 2003.

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these exceedingly high relative risks exist at doses far below the dose estimates of Dr. Sawyer and Mr. Kaltofen. The authors of Hayes 1997 demonstrated a 3.2 relative risk of contracting AML at just 6.7 ppm-years. Similarly, Gray 2001 showed a 5.71 relative risk for contacting AML at exposure levels of 0.8 to 4.8 ppm. This same study showed a 4.1 relative risk for leukemia at just 1.44 ppm-years and a 4.7 relative risk at just 2.78 ppm-year. Likewise, Glass 2003 showed a 7-fold increased risk of leukemia for at more than 8 ppm-years, causing the authors to conclude that the "risk of leukemia was increased at cumulative exposures above 2 ppm-years and with intensity of exposure of the highest exposed job over 0.8 ppm." Finally, the PADOH 2000 study demonstrated a 5.56 relative risk of contracting AML from no more than 2 ppm-years of benzene resulting from exposure to gasoline fumes.

The combined weight of these studies and the universal acceptance that benzene is capable of causing the disease Mr. Henricksen developed amply demonstrate Mr. Henricksen's cumulative

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[62] Terry 2005.

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exposure to more than 8 ppm-years of benzene from ConocoPhillips' gasoline was a cause of his AML.

5. Dr. Frank Gardner's Opinions Are Reliable and Admissible and The Opinion of Mr. Henricksen's Treating Physician that Mr. Henricksen's AML was caused by Benzene Exposure is Uniquely Reliable

Moreover, this evidence that Mr. Henricksen's cumulative exposure to benzene was far in excess of the dose required to double his risk of AML demonstrates the flaw in ConocoPhillips' motion to exclude Dr. Frank Gardner. Contrary to ConocoPhillips' contention, Dr. Garnder no where asserts that a particular AML may be caused at any dose of benzene.[63] Instead, based on his knowledge of the scientific evidence, Dr. Gardner correctly noted that benzene, including benzene from gasoline, causes AML at extremely low doses. Moreover, Dr. Gardner correctly assumed that, as a bulk fuel transporter, Mr. Henricksen would suffer extraordinarily high exposures as part of his job. Unlike the cases cited by ConocoPhillips in which no dose estimate was

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[63] ConocoPhillips also argues that Dr. Gardner's opinion should be struck because there is no epidemiological evidence that gasoline causes AML. Because the fallacies of this argument are addressed elsewhere in the brief, they will not be repeated here.

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was ever provided,[64] here Dr. Gardner's estimation that Mr. Henricksen's exposure was sufficient to cause disease was verified by two highly qualified experts.

Essentially, ConocoPhillips argues that Dr. Gardner should be excluded for making the assumption that Mr. Henricksen's exposure was sufficient to cause AML based on his extensive background and knowledge, even though that assumption ultimately proved to be true when tested by the other retained experts in this case. ConocoPhillips' argument for excluding the causation testimony of Mr. Henricksen's diagnosing physician, Dr. John Caton. In his deposition, Dr. Caton testified that he believed Mr. Henricksen's AML was caused by his exposure to benzene. As with Dr. Gardner, ConocoPhillips faults Dr. Caton's opinion because he did not have a specific dose calculation before reaching his opinion. However, Dr. Caton's opinion is not based on a specific cumulative dose calculation, but on the universal knowledge that gasoline contains benzene, benzene causes AML at very low doses, and tanker truck drivers such as Mr. Henricksen are exposed

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[64] *See, e.g., Castellow v. Chevron USA*, 97 F. Supp. 2d 780, 791-92 (S.D. Tex. 2000).

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exposed to high levels of benzene in their work. It is unclear what portion of *Daubert* that ConocoPhillips relies on for its assertion that the Court should exclude testimony based on assumptions that ultimately prove to be true.

VI. Summary Judgment

In its motions for summary judgment, ConocoPhillips again argues that Plaintiffs cannot produce any evidence of general or specific causation. As amply demonstrated above, ConocoPhillips is wrong in this contention. There is virtually no doubt in the scientific community that benzene causes AML, and there is no dispute that ConocoPhillips's gasoline contains gasoline. ConocoPhillips' argument that there is no evidence that gasoline causes AML is a red-herring that should be rejected by the Court.

Additionally, for ConocoPhillips to prevail on either its *Daubert* motions or its motions for summary judgment, it must convince the Court to make an impermissible factual finding. At most, ConocoPhillips' briefing establishes that qualified, credible experts disagree. Under the *Daubert* standard, FED. R. CIV. P. 56, and the 7th Amendment, these factual disputes remain the province of the jury. The Court should not

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supplant the role of the jury by resolving which expert's testimony is correct.

In short, because the general and specific causation testimony of Plaintiffs' expert witnesses is credible, ConocoPhillips is wrong in its contention that Plaintiff can produce no evidence to support the claim that Mr. Henricksen's AML was caused by his exposure to ConocoPhillips' gasoline. The testimony of these experts is some evidence with regard to both general and specific causation. Because a fact issue remains on these issues, both of ConocoPhillips' motions for summary judgment must be denied. FED. R. CIV. P. 56.

VII. Conclusion and Prayer

For the foregoing reasons, Plaintiffs respectfully request that the Court deny each motion to exclude expert testimony and each motion for summary judgment filed by ConocoPhillips. Plaintiffs also request all other relief to which they are entitled.

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DATED this 13th day of June, 2008.

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CERTIFICATE OF SERVICE

I hereby certify that on this date I electronically filed the foregoing document with the Clerk of Court using the CM/ECF system, which will send notification of such filing to the following attorneys of record who are CM/ECF participants:

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